AD-759 510

QUANTITATIVE INDICIES OF THE SUOCK SYNDROME

Benjamin W. Zweifach

California University

Prepared tor:

Army Medical Research and Development Center April 1973

DISTRIBUTED BY:



U. S. DEPARTMENT OF COMMERCE 5285 Port Royal Road, Springfield Va. 22151

QUANTITATIVE INDICIES OF THE SHOCK SYNDROME

FINAL REPORT

B. W. Zweifach

AMES-Bioengineering
The University of California, San Diego
La Jolla, California 92037

DDC PROFINIE MAY 4 SEE OUSUSIO ISU

April 1973

Supported by

U.S. ARMY MEDICAL RESEARCH AND DEVELOPMENT COMMAND Washington, D. C. 20315

CONTRACT NO. DA DA 17-67-C-7001

Approved for public release; distribution unlimited

The findings in this report are not to be construed as an official Department of the Army position unless so designated by other authorized documents

NATIONAL TECHNICAL INFORMATION SERVICE

(UNCLASSIFIED)

and classification of title body of sharest and ordered							
AMES-Sicengineering	uity classification of title, holy of obstract and indexing annotation must be of ACTIVITY Corporate author) Dengineering		2a. REPORT SECURITY CLASSIFICATION				
University of California, San Diego La Jolla, California 92037	iversity of California, San Diego						
Quantitative Indicies of the Shock Syndro	ome						
4. DESCRIP IVE NOTES (Type of report and Inclusive dates) Final Report, 1 July 1956 - 30 September	1973						
5. AUTHOF () (First name, middle initial, last name)							
B. W. Zweifach							
April 1973	70. TOTAL NO 0	FPAGES	76. NO. OF REFS				
62. CONT-ACT OR GRANT NO DADA17-67-C-7001	98. ORIGINATOR	98. ORIGINATOR'S REPORT NUMBER(\$)					
b. PROJECT NO.							
c.	95. OTHER REPORT NO(3) (Any other numbers that may be assigned this report)						
d.							
Approved for public release; distribution	n unlimited						
11. SUPPLEMENTARY NOTES	US Army Medical Research and Develop- ment, Command, Washington, DC 20314						
13. ABSTRACT							
Two forms of experimental shock were inv	estigated - h	emorrhagic	and bacterial				
endotoxemia - in the dog, cat and rabbit	. In addition	n to measu	rement of central				
arterial pressure, central venous pressu	re, heart rat	e, respira	tory rate, plasma				
pU2, pCO2 and pH, blood hematocrit, and	plasma colloi	d osmotic	pressure,				
observations were carried out on the mic	rocirculation	of the me	sentery and				
omer cum.							

	ROLE	₩ T	ROLE	w T	ROLE	
experimental shock				-		
hemorrhagic shock		,			! 	
bacterial endotoxemia						i
					:	
arterial pressure		}	,			
				٠		
		<u>.</u>				
·		}	İ			
·	ļ					
	ļ					
		<u> </u>				
•				ĺ		
		}				
			<u> </u>	Ì		
,						
·			İ	}		
			•	Ì		
				.		
		}				
		ļ				
					ļ	
	ļ	1				
				1		
	<u> </u>			<u> </u>	1	

Two forms of experimental shock were investigated - hemorrhagic and bacterial endotoxemia - in the dog, cat and rabbit. In addition to measurement of central arterial pressure, central venous pressure, heart rate, respiratory rate, plasma pO₂, pCO₂ and pH, blood hematocrit, and plasma colloid osmotic pressure, observations were carried out on the microcirculation of the mesentery and omentum. At the microvascular level measurements were made of micropressure, flow, fluid exchange and vascular permeability.

- (1) It was found that measurements of colloid osmotic pressure of the plasma ($\pi p \ell$) during the progression of circulatory insufficiency was a reliable index of the state of the individual. Compensation was associated with hemodilution and a lowering of $\pi p \ell$ by some 15-20%. This level was maintained for several hours, after which $\pi p \ell$ levels tended to rise. The latter phenomenon was accompanied by evidence of increased vascular permeability in the splanchnic region. The i.v. injection of a bolus of concentrated albumin (25%) also served as an index of the state of the animal. In normal and compensated individuals, such a bolus led to uptake of fluid and restoration of control levels of $\pi p \ell$ within 15-20 minutes. In decompensated animals, such an injection was not associated with fluid uptake and plasma c.o.p. levels remained elevated.
- (2) Experiments in which partial resection of the intestines was carried out, it could be shown that hemodilution (as measured by $\pi p \ell$ levels) was suppressed or absent. Hence, the major source of tissue fluid for the vascular system would appear to be the splanchnic viscera, in particular the intestinal tract. Additional attention should be given to fluid replacement therapy by this route.

- (3) A membrane osmometer was designed and tested which could be used as a clinical instrument. The unit is small, has a simple temperature control and provides readings within 2-3 minutes for fluid samples as small as 0.5 ml.
- (4) The measurement of micropressures and their distribution in the microcirculation during the shock syndrome made it possible to distinguish between vascular and blood derrangements. It was found that microvascular decompensation was the primary defect following hemorrhage, whereas there was some evidence of red blood cell aggregation and poor distribution of blood in the capillary network following lethal doses of bacterial endotoxins.
- (P_c) at a fixed level served as an index of vascular behavior. Periods of hypotension (below 50 mm Hg) in excess of two hours led to a progressive impairment of such vascular regulation. An ominous prognostic feature was the appearance of fluctuations in P_c in an upward and downward erratic course. This type of regulation is believed to represent a myogenic type of response of the smallest precapillary vessels.
- (6) In view of the need to balance hydrostatic pressures with plasma colloid osmotic pressures to maintain blood-tissue fluid balance, various volume replacement measures during hemorrhagic shock were evaluated both by following plasma c.o.p. levels and by observing fluid exchange in the microcirculation. A combination of balanced electrolyte infusion followed by the slow administration of 25% albumin was highly effective in restoring both of these features.
- (7) Studies on the regional distribution of the circulation after fatal endotonemia indicated a defective splanchnic adjustment within 90 minutes

after the initial insult.